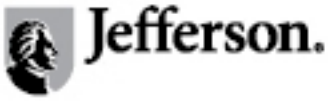

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Functional recovery of untreated human immunodeficiency virus-associated Guillain-Barré syndrome: A case report

Adam L. Schreiber
Jefferson Medical College of Thomas Jefferson University, Department of Rehabilitation Medicine, Philadelphia, PA, Adam.Schreiber@jefferson.edu

John W. Norbury III
Physical Medicine and Rehabilitation and Thomas Jefferson University Hospital; Department of Physical Medicine and Rehabilitation, Brody School of Medicine of East Carolina University, Greenville, NC, John.Norbury@jefferson.edu

Eduardo A. De Sousa
Department of Neurology, Division of Neuromuscular Disorders, at Jefferson Medical College of Thomas Jefferson University; Assistant Professor in the Department of Neurology, Division of Neuromuscular Disorders, University of Oklahoma - College of Medicine, Oklahoma City, OK

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Title Page

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Authors:
Adam L Schreiber, DO MA[1]
John W Norbury III, MD[2]
Eduardo A De Sousa, MD[3]

Affiliations:

1. Assistant Professor, Jefferson Medical College of Thomas Jefferson University, Department of Rehabilitation Medicine, Philadelphia, PA.

2. At the time of writing, JWN was a Resident Physician in Physical Medicine and Rehabilitation and Thomas Jefferson University Hospital and is now Clinical Assistant Professor in the Department of Physical Medicine and Rehabilitation, Brody School of Medicine of East Carolina University, Greenville, NC.

3. At the time of writing, EAD was an Assistant Professor in the Department of Neurology, Division of Neuromuscular Disorders, at Jefferson Medical College of Thomas Jefferson University and is now Assistant Professor in the Department of Neurology, Division of Neuromuscular Disorders, University of Oklahoma - College of Medicine, Oklahoma City, OK.

Address correspondence to:

Adam L. Schreiber, DO, MA
25 S 9th Street
Philadelphia PA 19107
Phone : 215-955-4206 Fax: 215-955-0861
Email: adam.schreiber@jefferson.edu

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Abstract

HIV-associated Guillain-Barré Syndrome is a well-documented phenomenon, typically occurring at seroconversion. GBS may result in functional impairment treated with a combination of medications, plasmapheresis, and rehabilitation. The quantified functional recovery of HIV-associated GBS with or without HIV treatment is not well-described. Utilizing serial FIM scoring, we describe a patient's recovery from HIV-associated GBS after treatment with IVIg and acute inpatient rehabilitation without HIV treatment.

Key words: Guillain-Barré Syndrome, human immunodeficiency virus, rehabilitation, Demyelinating Diseases

Introduction

Standard Guillain-Barré Syndrome (sGBS) is an autoimmune disorder characterized by sudden onset of weakness with evidence of demyelination through electrodiagnosis.[1] This condition often requires pharmacological and/or plasmapheresis treatment. After treatment, 40% of sGBS patients require inpatient rehabilitation.[2] The most rapid improvement is the first 6 months.[3] sGBS recovers during the first year of onset, motor improvements at 18 months, but motor and sensory impairments are detectable in more than 50% of patients at two years.[4] Functional Independence Measure (FIM™) is a proven tool to monitor rehabilitation progress of sGBS.[5]

There are several peripheral neuropathies associated with HIV.[6] HIV-associated GBS (hGBS) is well documented.[7,8,9] which may occur during the entire course of the disease, but typically occurs at seroconversion,[10,11,12] but may Quinn's states "neurological impairment [from HIV] is generally self-limited; most patients become asymptomatic in 1 to 4 weeks, although persistent neurologic deficits have been described."[13] Recent literature describes prolonged recovery of HIV-related chronic neurologic conditions. [10] Verma speculates that the clinical course and response to pharmacologic treatment for GBS in HIV-seropositive and seronegative patients is similar[14], but the functional recovery of hGBS with or without HIV treatment is not well described. We describe a case of hGBS without HIV treatment with subacute recovery.

Case report

A 45-year-old woman presented with left sided weakness after 10 days of headache with nausea and vomiting and an inability to ambulates. CT and MRI of the brain and spine were unremarkable. She refused lumbar puncture. Electrodiagnostic studies revealed demyelinating sensorimotor polyneuropathy without denervation. Diagnosed with Guillain-Barré Syndrome (GBS), she was treated with IVIG, and discharged to inpatient rehabilitation.

On admission, her initial exam revealed areflexia with intact sensation to light touch and pinprick. There were no symptoms of dysautonomia but dysesthesias were present in her bilateral hands. Manual muscle testing revealed grade 5-/5 humeral abductors, 4/5 elbow flexors/extensors, wrist extensors, 3+/5 flexor digitorum profundi, 2/5 dorsal interossei, 3/5 hip flexors, 4/5 knee extensors, and 4/5 ankle dorsi/plantar flexors bilaterally.[15] Functional Independent Measure (FIM^{TM}) scores showed functional deficit requiring moderate assistance for self care, minimal assistance for sphincter control, and dependence for ambulation (Table 1). She initially made gains but then experienced progressive weakness and decline in many functional domains (Table 1).

Work-up included a second electrodiagnostic study approximately 1 month after initial study, revealing more evidence of a demyelinating polyneuropathy (Table 2): severe distal motor latency prolongation and abnormal distal temporal dispersion (duration >9.0 ms) in motor nerves. Left peroneal and right median abnormal proximal temporal

dispersion. Sural responses were present with attenuated amplitudes. No conduction blocks or demyelinating F-waves (absent F-waves with relatively normal CMAP amplitude, or severely prolonged F-wave minimal latencies) were noted. EMG revealed denervation suggestive of secondary axonal injury. Lumbar puncture had elevated protein 240mg (normal value 15-55) and cell count of 39WBC & 8RBC with normal glucose 53mg/dL (normal value 40-70). Because of elevated CSF cell count and protein, further work revealed positive HIV ELISA and confirmed by western blot. CD4 count of 334cells/ml (normal value 410-1590) and viral load 394,000copies/ml (normal value <400).

Rehabilitation was reinitiated without further pharmacological treatment for GBS or HIV. Due to concern for compliance, infectious disease consultant did not treat HIV. One month after readmission she displayed functional improvement at supervision for most self care domains, independence with sphincter control, modified independent for mobility at a wheel chair level (Table 1). Her exam on discharge revealed symmetric 1+ reflexes with sensation intact to light touch and pinprick. Manual muscle testing revealed 4+-5/5 strength in all muscle groups. Three months after discharge and five months after onset of hGBS, she had complete functional recovery; independent in all FIM^{TM} domains including ambulation without assistive device (see Table 1). She was lost to further follow-up.

Discussion

We present a case of GBS occurring as the first manifestation of HIV infection and her functional recovery after IVIG and inpatient rehabilitation which suggests that hGBS

patients may make full subacute recovery without the initiation of HAART therapy. To our knowledge, there are no reports of acute seroconverted hGBS rehabilitation recovery without HIV treatment. This may add to the controversy in the literature regarding hGBS treatment with HAART with both relapse[16] upon discontinuation or cause due to drug side effect or of HAART or potentially cause via immune reconstitution syndrome.[17]

Neurologic impairment related to HIV are recognized have prolonged recovery at best.[13, 18] Our case presents hGBS taking 5 months full functional recovery. This is timely as compared to the described recovery of sGBS and confirms Verma's speculation.[14]

Limitations of this manuscript include the use of FIM™ scoring to quantify functional recovery, as there are other tools to evaluate recovery of GBS patients.[19] Additionally, she was lost to follow-up to monitor for function over a longer period of time.

Conclusion

We present a case of Guillain-Barré Syndrome as the initial clinical manifestation of HIV infection. With standard GBS treatment, without HIV treatment, the patient made similar functional recovery, quantified by FIM™ scoring, that is described for sGBS. Further research is needed on the recovery of HIV-associated GBS with various treatment strategies so physicians can better prognosticate functional recovery.

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